

Ecological and social constraints on conspecific brood parasitism by nesting female American coots (*Fulica americana*)

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Summary

1. In a population of American coots (*Fulica americana*) breeding in central British Columbia, Canada, some females pursued a reproductive strategy that combined nesting with laying parasitic eggs in the nests of conspecifics. To understand why only one quarter of the nesting females laid parasitically, I examined social and ecological factors that could potentially constrain nesting females from engaging in brood parasitism.
2. Variation among females in each of three prerequisite conditions essential for adaptive parasitism to occur – host availability, access to host nests and a benefit to allocating eggs to parasitism – helps explain why not all females are parasitic.
3. Due to limited spatiotemporal patterns of parasitism, an estimated 23% to 39% of non-parasitic females completely lacked potential hosts to parasitize when they began breeding. In theory, females could avoid host limitation by delaying breeding to wait for hosts, but timing of breeding comparisons did not support this idea. Relative to the vegetation density on their territories, parasites and non-parasites began laying eggs on the same date but, due to the time taken to lay parasitic eggs, parasites initiated their own nests later.
4. Brood parasitism was less likely to occur between dyads of females where the potential host female was substantially larger than the potential parasitic female, which suggests that antagonistic social interactions between hosts and parasites may constrain some females from parasitism.
5. Comparisons of two classes of non-parasitic females, those with and without hosts, revealed smaller clutch sizes in the former, suggesting that limited fecundity may have constrained some of these females from laying parasitic eggs. Additionally, brood parasites were older and laid more total eggs than non-parasitic females and, among parasites, older females laid more parasitic eggs. Together, these patterns suggest that variation in egg-laying capacity may determine whether females benefit from allocating eggs to parasitism.
6. The large number of correlates of parasitism indicates that parasitism by nesting females is a conditional reproductive tactic, not part of a stochastic mixed evolutionary stable strategy.

Key-words: conspecific brood parasitism, clutch size, host limitation, host–parasite interaction, timing of breeding.

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Introduction

In birds and insects, alternative female reproductive behaviours are widespread in the form of conspecific brood parasitism, whereby females lay eggs in the nests

of conspecifics but do not provide care for the young (Brockman, Grafen & Dawkins 1979; Yom Tov 1980; Eadie, Sherman & Semel 1998). This behaviour has now been documented in over 200 species of birds (Rohwer & Freeman 1989; Eadie *et al.* 1998; Davies 2000) and two basic contexts have been identified, each with different constraints and trade-offs. In some populations, most parasitism is by non-nesting females

who are unable to breed due to poor physical condition, nest site limitation, or territory saturation (e.g. Emlen & Wrege 1986; Lank *et al.* 1989). In other species, parasitism is primarily by females with nests of their own (e.g. Gibbons 1986; Jackson 1993; Lyon 1993a; McRae & Burke 1996). Parasitism by nesting females is particularly interesting because these females must divide time and effort between parasitism and parenting, potentially giving rise to trade-offs between the two reproductive tactics (Jackson 1993; Lyon 1993a, 1998; Yamauchi 1993).

Several studies have sought to explain how nesting females benefit from laying some of their eggs parasitically (Gibbons 1986; Brown & Brown 1989; Sorenson 1991; Jackson 1993; Lyon 1993a; McRae 1998; Sandell & Diemer 1999; Ahlund & Andersson 2001), or in some cases, if there is a benefit at all (Semel & Sherman 2001). In most populations, however, only a minority of nesting females engage in parasitism, and demonstrating how these females benefit from parasitism is insufficient to fully explain individual variation in parasitic behaviour. A complete understanding of parasitism should not only be able to explain why some females are parasitic, but also why other females are not. Such a level of understanding is currently lacking for any conspecific brood parasite.

Here, I explore ecological and social factors that can explain the variation in parasitic tendencies among nesting female American coots (*Fulica americana*), monogamous marsh-nesting members of the family Rallidae. During my 4-year study in central British Columbia, one quarter of some 400 nesting females engaged in parasitism (Lyon 1993a). Elsewhere, I showed that these females were able to lay more eggs than they could raise in their own nests, due to the constraints of post-laying parental care, and parasitism allowed them to bypass these constraints and increase their total production of offspring (Lyon 1993a, 1998). Although this explanation accounts for how parasitic females benefited from parasitism, it does not explain why three quarters of the nesting females in the population did not lay any parasitic eggs. My goal here is to understand this variation. I first develop a simple conceptual framework for investigating factors that could constrain nesting females from engaging in brood parasitism and I then evaluate these constraints for female American coots. Because my study is observational and any support for the hypothesized constraints is thus correlational, I placed special emphasis on conceiving and testing any alternative hypotheses that could also account for the patterns predicted by each of the constraint hypotheses.

CONCEPTUAL FRAMEWORK FOR EXPLORING CONSTRAINTS ON PARASITISM

Three general prerequisites must all be met for a nesting female to successfully pursue adaptive brood parasitism: (i) she must have host nests available to

parasitize, (ii) she must be able to find and gain access to at least one of those nests, and (iii) she must benefit from allocating some of her eggs to parasitism. The absence of any one of these prerequisite conditions would be sufficient to prevent parasitism; thus, different females may be prevented from laying parasitically for different reasons. These hypotheses and their predictions are summarized in Table 1. Below, I outline in more detail the logic behind each of these conditions and identify predictions to test whether each condition constrains some female American coots from laying parasitically.

Host availability

Some females may be prevented from laying parasitic eggs because they lack hosts to parasitize. Host availability for a nesting female will depend on both the spatial scale of parasitism (distance over which parasitism is feasible) and the temporal relationship between the parasite's and host's reproductive cycles. Empirically, one can determine the pool of potential hosts available to a given female based on the observed spatial and temporal patterns of actual host use by nesting parasites in a population (Emlen & Wrege 1986; Brown & Brown 1991; Lyon 1993b).

Under some conditions, host availability and limitation will be influenced by timing of breeding decisions. For example, because female coots lay parasitically before they begin nesting and because parasitism is limited to a radius of two territories, the earliest nesting female in each social neighbourhood lacks potential hosts to parasitize when she begins to lay eggs because none of her neighbours have nests yet (Lyon 1993b). In theory, these host-limited females could bypass host limitation simply by delaying nesting to wait for hosts to become available. The females that then remain without potential hosts would be those females that were constrained from parasitism for reasons other than host limitation, and that gain no benefit from waiting for hosts. This scenario can be rejected by demonstrating that parasites do not breed later than non-parasites. Clearly, a full understanding of the importance of host limitation requires an understanding of the factors that influence both the timing of breeding, and its flexibility.

Social interactions and access to host nests

Some females with potential hosts available may be prevented from laying parasitic eggs in those host nests because (i) the potential parasites cannot find the host nests, (ii) the nests are defended too aggressively by the hosts (McRae 1996; Sorenson 1997), or (iii) dominance relationships are so clear that the potential parasite does not even attempt to gain access to the host nest. If nest discovery plays a role, then unparasitized nests should be better hidden than parasitized nests. If host nest defense or dominance relationships between hosts

Table 1. Hypothesized factors that could constrain nesting female birds from engaging in adaptive brood parasitism, and the predictions of each hypothesis. Any one of these factors would be sufficient to constrain brood parasitism and different females in a population may be constrained for different reasons

Hypothesis	Predictions
1. Host constraint	1. Due to spatiotemporal patterns of brood parasitism, some non-parasitic females have no hosts available to parasitize when they begin to breed <i>and</i> they do not alter their timing of breeding to wait for hosts to become available
2. Nest defence by hosts prevents some females from gaining access to host nests	2. Relatively small or subordinate females are less likely to be parasitic; parasitic females are larger or more dominant than non-parasitic females, or hosts are physically smaller or less dominant than non-parasitized birds
3. The costs of allocating eggs to parasitism outweigh the benefits because:	
i. Non-parasites are less fecund (total potential fecundity) than parasites	i. Non-parasites can lay fewer total eggs than parasites; non-parasites show signs of low fecundity like reduced clutch size or reduced renesting potential
ii. Non-parasites live on territories subject to higher nest predation	ii. Parasites and non-parasites have the same total fecundity, but non-parasites suffer a higher risk of nest predation, and benefit from allocating any extra fecundity to renesting
iii. Non-parasites have higher quality breeding situations that favour larger parental clutch sizes	iii. Parasites and non-parasites have the same total fecundity, but non-parasites have better breeding situations (i.e. can fledge more chicks) that favor allocating eggs to a larger parental clutch size instead of parasitism
iv. The time costs of laying parasitic eggs exceed any fitness benefits except for females who must wait for mates of territories	iv. Parasites are constrained from breeding for reasons external to parasitism and lay parasitically while waiting; for other females, parasitism would delay breeding and the costs of this delay outweigh the benefits of laying parasitically

and parasites prevent some females from laying parasitically, then morphological characteristics that affect dominance and aggression are predicted to differ between birds at parasitized and unparasitized nests. Characteristics of parasitic females could also influence access to host nests; if so, attributes of parasitic females should differ from those of non-parasitic females. Finally, as dominance is a function of encounters between specific individuals, demonstrating that the occurrence of parasitism is affected by the *relative* attributes of females in specific dyads would be particularly powerful.

Variation in the benefits of allocating eggs to parasitism

Parasitism by nesting females can be viewed as a clutch size problem, whereby a female must allocate eggs between her own nest and parasitism (Lyon 1998). Allocating eggs to parasitism only pays when the fitness gain of laying an egg parasitically exceeds the benefit that would be gained from laying the egg in the parental nest. In species like coots where parasitic eggs yield very low fitness returns (Lyon 1998), the clutch sizes where it pays a female to switch allocation to parasitism instead of the the female's own nest can be very large. Accordingly, some females may forgo parasitic laying not because host availability or access is limiting but because, due to limited fecundity, they gain less from allocating eggs to parasitism than they gain from allocating the eggs to their own nest. In other words, these females cannot even lay their optimal

parental clutch size, let alone allocate additional eggs to parasitism.

Several factors could influence the allocation of eggs between parasitism and the parental nest, so that some females are fecund enough to benefit from parasitism, while others are not. First, females may vary in their ability to produce eggs (i.e. in total fecundity), due to differences in foraging experience or territory quality. This hypothesis predicts that females constrained from parasitism by their limited fecundity should have smaller than average clutch sizes. Second, females might not differ in total fecundity but, instead, vary in the optimal clutch size they should lay in their own nest, due to variation in territory or parental quality that affects food availability for chicks. Depending on the total number of eggs that can be laid, perhaps only females with small optimal clutch sizes have additional eggs to allocate to parasitism. This hypothesis predicts that parasites have worse breeding situations (lower clutch size *combined with* worse fledging success) than non-parasites. Third, females may differ in their risk of nest predation. If females have reliable cues to assess these risks, birds on low-risk territories may be able to allocate eggs to parasitism, while those on high-risk territories might benefit instead from allocating limited eggs to replacement clutches. This hypothesis predicts that brood parasites suffer lower rates of nest predation than non-parasitic females.

One other trade-off may affect the net benefit of parasitism to a female, namely time trade-offs (Gibbons

1986). In many birds, reproductive success declines with season (Klomp 1970), and if brood parasitism delays the parasite's own nest initiation, parasitism may incur temporal costs. If these costs exceed the benefits of parasitism, parasitism may only benefit females who are constrained from breeding because they have to wait for their territory or mate to reach a state suitable for breeding (Gibbons 1986). As these females are delayed from breeding irrespective of whether they lay parasitic eggs, the time costs of parasitism do not affect the net benefit they derive from parasitism. This hypothesis predicts that (i) the time costs incurred by laying parasitically exceed the fitness benefits and (ii) the parasites are prevented from breeding early by factors external to brood parasitism (e.g. territory or mate attributes).

Methods

STUDY AREA AND ANIMAL

I studied brood parasitism by nesting females from 1987 to 1990 at three sites within 60 km of each other near Riske Creek in central British Columbia, Canada. In 1987, I studied coots on several wetlands at Riske Creek but, due to a drought, moved to other sites for the duration of the project. The two other sites, Jaimeson Meadow near Big Creek and the Chilco West cluster of wetlands near Hanceville (Kloe Lake, Jone's Lake, Pond S5) were managed by Ducks Unlimited Canada and water levels were maintained at high levels from 1988 to 1990. Hardstem bulrush (*Scirpus acutus*), the dominant emergent plant at all wetlands, was limited to a shoreline strip on most wetlands but sparse patches of bulrush grew in the middle of both Kloe Lake and Jaimeson Meadow, providing nesting cover for coots away from the shoreline.

At these sites, coots are migratory and my banding studies show that adult philopatry is very rare (unpublished data), hence I studied different individuals each year. Coots are monogamous and males share in all aspects of reproduction including nest-building, incubating, feeding and brooding the chicks, and defending the territory (Gullion 1953). Coots defend their all-purpose territories throughout the entire reproductive cycle and families remain exclusively on the parental territory until chicks are about 50 days old. The chicks are semi-precocial and mobile, but depend critically on their parents for food for at least 10 days (Lyon 1993a). Nest predation was frequent on most wetlands and some females laid up to three replacement clutches. Virtually all pairs were single-brooded.

CENSUSING NESTS, DETECTING PARASITISM AND IDENTIFYING PARASITIC FEMALES

I searched for new nests and monitored known nests every 1–2 days. Virtually all nests were discovered early in the laying period and because territorial birds were

so conspicuous and their territories so small, I am confident that I found all nests on each wetland. On each nest visit, all new eggs were numbered with an indelible felt pen, and these numbers were maintained until hatching. I used three standard criteria to determine when parasitism had occurred (e.g. Brown 1984; Gibbons 1986; McRae & Burke 1996): (i) two or more new eggs per day, (ii) new eggs two or more days after clutch completion and, rarely, (iii) variation in egg features. Birds are unable to lay more than one egg per day, so the first criterion alone provides convincing evidence that parasitism has occurred. In virtually all instances, timing of laying (the first two criteria) was used to identify parasitized nests. At the focal wetlands (see below) 66% of the 179 nests were checked daily (Jaimeson 1988, S5 1989), while 33% were checked every second day (S5 1990), so the timing of laying criteria would have detected virtually all cases of parasitism (Lyon 1993a). Once instances of parasitism were detected based on timing of laying, egg features were then used to determine which of the new eggs was the parasitic egg and which was the host egg. Additional information on the factors affecting the accuracy of the three criteria used to identify parasitized nests can be found in Lyon (1993a).

Striking variation among females in egg features (Arnold 1990; Lyon 1993a) made it possible to visually match parasitic eggs to the females that laid them when the parasites also had nests of their own. I visually compared eggs in the field, examining features such as shape, spot colour, spot size, spot density and background colour. Identification of parasites was also supplemented with information about females' laying schedules: females who laid eggs in their own nests on the night a focal parasitic egg was laid were ruled out as potential parasites. Two thirds of all parasitic eggs ($n = 591$) were attributed to nesting females, and one quarter could not be attributed to any nesting females on the wetland and were assumed to have been laid by non-nesting females (Lyon 1993a). The few cases (6% of eggs) where I suspected, but could not confirm, that specific females had laid parasitically were excluded from analyses based on parasitic status. The accuracy of my ability to both identify parasitized nests and parasitic females by these field techniques has been verified by discriminant function analysis based on egg features (Lyon 1993b) and by DNA fingerprinting (Lyon, Hochachka & Eadie 2002).

ASSESSING POTENTIAL CORRELATES OF PARASITISM

Each year from 1988 to 1990, I chose a single focal lake to determine the temporal and spatial pattern of host use and availability, and to trap birds for morphological measurements; Jaimeson Meadow in 1988, Pond S5 in both 1989 and 1990. These two focal wetlands differed in three important aspects: (i) much more of the parasitism at Jaimeson Meadow was due to non-nesting

females than at Pond S5 (Lyon 1993a) (ii) the vegetation was considerably more dense at Pond S5 and (iii) 1988 was a drought year where breeding failed on unmanaged wetlands, and some aspects of reproduction differed when I compared the same wetland (Jaimeson) across years (e.g. lower clutch size, lower fledging success, and higher use of marginal habitat in 1988 relative to other years). Given these differences between the two wetlands, I conducted two sets of analyses: (i) pooled data for both focal wetlands and (ii) separate analyses for each wetland (Jaimeson in 1988; S5 for 1989 and 1990 combined). However, I only report the results of the separate comparisons when they differed from the pooled comparisons.

I examined several morphological characteristics of parasitized and unparasitized birds that could logically affect the outcome of social interactions between hosts and parasites, such as size, age and size of the frontal shield above the beak, a structure of particular interest because it functions as a signal of dominance in coots and gallinules (Gullion 1951; Petrie 1988). Birds were trapped at their nests during late incubation with automatic nest traps, were weighed, measured (tarsus, wing, length of the frontal shield) and fitted with a numbered neck collar. Following Petrie (1988), I measured frontal shield as the length of the upper mandible plus length of shield proper. Structural size contributes to this measure so I calculated relative shield size as the residual shield size that remained after the influence of body size (tarsus) was removed with regression. Leg colour is highly correlated with age in American coots and thus serves as an index of age (Gullion 1952; Crawford 1978). I was unable to classify birds clearly into the age categories designated by Crawford's (1978) ageing scheme because many individuals had colours diagnostic of more than one age class so, following Arnold (1990), I modified Crawford's scheme to give these birds intermediate values.

To control for differences among years in the timing of breeding, the day the first egg was laid in the population each year was designated as day zero and dates for nest initiation or parasitic egg-laying are expressed as number of days after day zero ('day of season'). Chicks were considered to have 'fledged' if they survived 30 days after hatching because very little mortality occurred between 30 and 50 days, at which point chicks began leaving the parental territory. When comparing fledging success, I did not include birds who failed to hatch eggs (i.e. birds who did not re-nest or whose nests were repeatedly depredated) because I was interested in characteristics of territories or parents that affected post-hatching survival. Nest predation was analysed in a separate comparison.

I quantified two territory characteristics that could affect the occurrence of parasitism, territory size and the density of vegetation cover. I measured territory size by marking the locations of territory borders with flagging tape whenever fights between neighbours were observed. On Pond S5 birds defended strips of

vegetation along the shoreline and territory size was measured as the length of shoreline defended. At Jaimeson Meadow, I measured territory size in terms of area because many females nested away from shore in the centre of the wetland. To convert territory area to a linear metric similar to the measures at Pond S5, I took the square root of territory area. Territory sizes differed between the two wetlands, so separate comparisons were done for each wetland.

I censused vegetation density on each territory late in the chick stage, when new vegetation growth was complete. The vegetation in each of four quadrants within a 10-m radius of the nest was visually ranked on scale of 1–7, and then averaged to yield a single vegetation density index for each nest. A rank of 1 signifies open water with at most a few strands of *Scirpus*, while a rank of 7 signifies solid dense growth with virtually no patches of open water. At some wetlands, independent indices were obtained by two or three observers and these were highly correlated (range of Spearman rank correlations for different pairs of observers, 0.89–0.98; mean of the six correlations 0.94), indicating that the ranking system was objective and consistent. Vegetation density differed between Jaimeson and Pond S5 so separate comparisons were done for each wetland.

HOST AVAILABILITY FOR PARASITES AND HOST VULNERABILITY TO PARASITISM

I determined the radius of potential hosts available to each female based on the observed spatial and temporal patterns of actual host use by nesting parasites (Lyon 1993b). Virtually all parasites laid their parasitic eggs immediately prior to initiating the clutches in their own nests, they parasitized birds within a radius of two territories and they laid eggs randomly with respect to hosts' laying periods (54% of 355 parasitic eggs) and incubation periods (46%; Lyon 1993b). Combining these spatial and temporal requirements, a potential host for a focal female was defined as any nest within a radius of two territories that contained eggs when the focal female laid her first egg of the year. For parasitic females, this first egg was typically laid parasitically, while for nesting females it was the first egg they laid in their own nests. Even though parasites laid eggs in both the host's laying and incubation periods, I conducted an additional analysis to ensure that the decision to lay parasitically *per se* was not affected the stage of the host's breeding cycle. It was not (unpublished data). Given that I sought to determine host availability based on what the parasites actually use, I therefore ignored host nesting stage (and value) when designating potential hosts.

In comparisons of attributes of parasitic and non-parasitic birds, I sought to increase statistical power by excluding from the analysis all birds that lacked potential hosts to parasitize. In other words, after I confirmed that host availability is an important constraint, I wanted to then examine the subset of birds for

which host availability was not a confound. To determine whether any conclusions from these analyses were affected by the criteria I used to define potential hosts, I repeated all analyses using two different criteria: (i) potential hosts restricted to a radius of one territory (since 85% of parasite–host dyads were immediate neighbours), not two, and (ii) potential hosts limited to birds early in incubation, rather than any bird whose nest contained eggs. Changing these criteria did not affect any of the conclusions.

I similarly sought to increase statistical power when assessing attributes of hosts. I excluded from comparisons of parasitized and unparasitized birds all pairs that were not *vulnerable* to being parasitized by any of their neighbours ($n = 20$ pairs). I did this with the assumption that the lack of parasitism on non-vulnerable birds was explained by their timing of breeding relative to their neighbours, not by potential host–parasite social interactions. Host vulnerability is just the converse of host availability: any nest that is a potential host nest to a given female is potentially vulnerable to being parasitized by that female.

Unless indicated otherwise, values reported with means are standard errors and probabilities are two-tailed probabilities.

Results

TEMPORAL AND SPATIAL PATTERNS OF HOST USE AND AVAILABILITY

Three related patterns indicate that host availability influences the occurrence of brood parasitism. First, parasitic females had more potential hosts available to them (2.96 ± 0.18 hosts, $n = 46$) than did non-parasites (2.08 ± 0.15 hosts, $n = 111$; Mann–Whitney U -test, $z = -3.27$, $P < 0.001$). Second, the probability that a female laid parasitically increased with the number of potential hosts available to her (Fig. 1a; Spearman correlation, $r_s = 1.0$, d.f. = 4, $P < 0.02$; 25% of the females with a single potential host were parasitic, compared to 50% with five or more potential hosts). Third, a substantial proportion of non-parasitic females had no potential hosts available to them when they began to lay (Fig. 1a; 23% of the 111 non-parasitic females). These were the earliest nesting females in each local neighbourhood who preceded all of their neighbours within a two territory radius and, as a result, had no potential hosts when they laid their first egg. Restricting the pool of potential hosts to a radius of one territory almost doubles the estimate of host limitation: 38.7% of the 111 non-parasitic females had no potential hosts available with a radius of one territory when they began breeding.

Because host availability depends on a female's timing of breeding relative to her neighbours, females that bred later in the season had more potential hosts than early breeding females (Fig. 1b; Spearman rank correlation between a female's first egg date and the

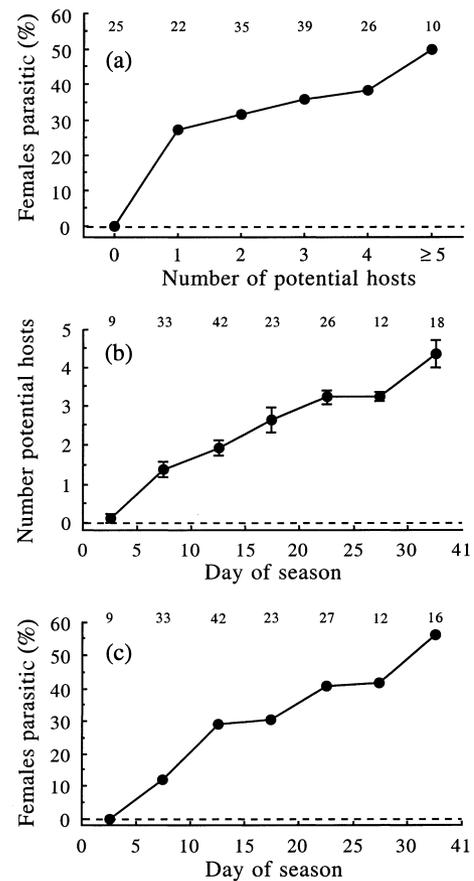


Fig. 1. Host availability and the frequency of brood parasitism. (a) The probability that females laid parasitically (proportion of females parasitic) in relation to the number of potential hosts available to them. (b) The mean number of potential hosts available to females in relation to the date when females laid their first egg of the year. Day of season is number of days after day zero, the day the first egg in the population was laid each year. Each 5-day interval includes the day on the left side of the 5-day interval. (c) Percentage of females initiating breeding in each 5-day interval that were parasitic. Numbers above data points indicate sample size.

number of potential hosts available to her; $r_s = 0.63$, $n = 157$, $P < 0.001$). The frequency of parasitism showed a similar pattern of seasonal increase (Fig. 1c), probably because females with more potential hosts are more likely to lay parasitically (Fig. 1a).

DO PARASITES WAIT FOR HOSTS?

Simply demonstrating that some females lacked potential hosts is not, of itself, convincing evidence for host limitation because potential parasites who lacked hosts when they were ready to breed may have delayed breeding until hosts became available. If this occurred, then the birds observed without hosts would simply be females that were constrained from parasitism for reasons other than host availability and who thus had no reason to wait for hosts. If parasites do delay breeding to wait for specific hosts, then parasites should begin breeding later in the season than non-parasites, all else being equal.

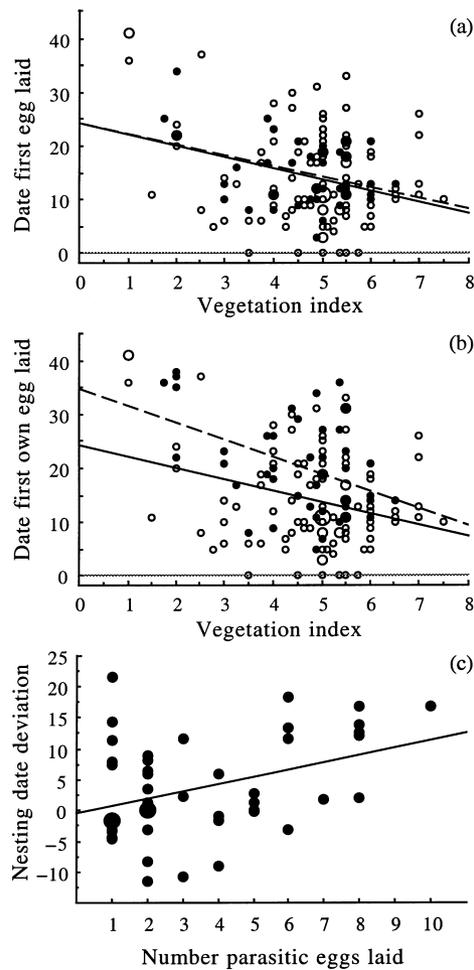


Fig. 2. Timing of breeding of parasitic females and non-parasitic females, relative to the vegetation density on their territories. (a) The date that females laid their first egg, which for parasites were parasitic eggs, and (b) the date that females laid the first egg in their own nests, in relation to the density of vegetation on their territories (index of 1 denotes sparse vegetation, index of 7 denotes dense vegetation). Lines are simple regression lines. Dates of first egg and first own egg differ only for parasitic females. Filled circles and dashed regression lines denote parasitic females, open circles and solid regression lines denote non-parasitic females. (c) Relation between the number of parasitic eggs laid by brood parasites and their delay in nest initiation; simple regression, $F_{1,42} = 6.76$, $P = 0.013$. Nesting delay was measured as the residual date from the regression of date of first egg vs. vegetation index for all birds. Large points indicate multiple samples with identical values.

Vegetation density predicts timing of breeding in coots (i.e. first egg of the year) and thus serves as a good external reference variable with which to compare timing of breeding of parasitic and non-parasitic females (regression of date first egg on vegetation index; $F_{1,290} = 57.3$, $P < 0.0001$, $R^2 = 0.17$, all wetlands studied 1988–90). Recall that parasitic females lay parasitically prior to nesting so their first eggs of the year are parasitic eggs. Relative to vegetation density, parasitic and non-parasitic females did not differ in the date they laid their first eggs (Fig. 2a; ANCOVA adjusted mean date first egg for parasites, day 15.1 ± 0.92 ($n = 44$); for non-parasites, day 14.2 ± 0.89 ($n = 103$); $F = 0.08$, $P =$

0.77). Parasites did, however, initiate their own nests later than non-parasites (Fig. 2b; ANCOVA adjusted date first own egg for parasites, day 20.5 ± 1.34 ($n = 45$); date for non-parasites as above; $F = 13.66$, $P = 0.0003$). The delay in initiation of parasites' own clutches relative to non-parasites can be explained by the time taken to lay parasitic eggs (Fig. 2c). The slope for the regression of number of parasitic eggs laid in relation to number of days delayed, $1.17 \text{ days egg}^{-1}$, is very close to a slope of 1.0 , the predicted value if each parasitic egg laid causes a one day delay in nest initiation. Thus, relative to the density of their territory vegetation, parasitic females began breeding at the same time as non-parasites, and there is no evidence that parasites delayed laying their first egg of the year to wait for hosts. Parasitism did, however, delay initiation of the parasites' own clutches, indicating that parasitism does affect timing of nesting and may involve temporal costs for the parasites.

CHARACTERISTICS OF HOSTS

Parasitized nests were not on territories with less vegetation cover (mean vegetation index 4.79 ± 0.15 , $n = 58$) than non-parasitized nests (4.68 ± 0.15 , $n = 80$); Mann–Whitney U -test, $z = 0.30$, $P = 0.76$). There were no significant differences between females at parasitized and non-parasitized nests in any of the morphological attributes I measured (Table 2). However, males at parasitized nests had significantly smaller frontal shields (Table 2; sequential Bonferroni adjustment of table-wide error rate based on four male trait comparisons, $P < 0.05$, Rice 1989). Further analysis by wetland showed that this frontal shield difference held only at Jaimeson Meadow in 1988. Moreover, parasitized males at this wetland were smaller than unparasitized males in two other morphological measures (wing: $t = 2.39$, $P = 0.03$; tarsus: $t = 2.0$, $P = 0.06$).

CHARACTERISTICS OF PARASITES

To determine whether access to host nests might also be influenced by characteristics of the parasitic female or her mate, I compared the attributes of parasites and non-parasites. Parasitic females did not differ from non-parasites in size, condition index or frontal shield size, but they were older than non-parasites (Table 3; sequential Bonferroni adjustment of table-wide error rate, based on five female trait comparisons, $P < 0.06$). The relation with age was strong at Pond S5 (both years combined; Mann–Whitney U -test, $z = -3.34$, $n = 15, 26$, $P < 0.001$) but non-existent at Jaimeson Meadow ($z = -0.40$, $n = 10, 11$, $P = 0.68$). Female age was also correlated with the total number of parasitic eggs laid by parasitic females (Spearman rank correlation, $r = 0.60$, d.f. = 24, $P < 0.02$). Subsequent analysis revealed this pattern was restricted to Pond S5 (Fig. 3, $P < 0.02$). Among parasites, older females also

Table 2. Morphological attributes of hosts and unparasitized birds. Only pairs vulnerable to parasitism were included in analyses (see text). Values are means \pm SE, with sample sizes in parentheses

	Host	Unparasitized	Statistic*	P
I. Females				
Relative frontal shield (mm)	0.16 \pm 0.22 (33)	-0.41 \pm 0.28 (33)	0.56	0.58
Mass (g)	521.3 \pm 8.2 (34)	510.0 \pm 5.9 (32)	1.11	0.27
Tarsus (mm)	63.4 \pm 0.31 (33)	63.6 \pm 0.35 (33)	-0.47	0.64
Age index (years)	1.74 \pm 0.13 (34)	1.46 \pm 0.11 (33)	-1.69	0.09
II. Males				
Relative frontal shield (mm)	-0.31 \pm 0.22 (38)	0.59 \pm 0.31 (29)	-2.43	0.018
Mass (g)	668.9 \pm 7.4 (38)	651.7 \pm 8.1 (30)	1.54	0.13
Tarsus (mm)	70.0 \pm 0.28 (38)	69.7 \pm 0.31 (29)	0.58	0.57
Age index (years)	1.70 \pm 0.09 (38)	1.75 \pm 0.14 (30)	0.00	1.00

*Mann-Whitney *U*-test (*z*) for Age index, *t*-test for all other attributes.

Table 3. Morphological attributes of parasites and non-parasites. Birds with no potential hosts to parasitize were not included in the analyses (see text). Values are means \pm SE, with sample size in parentheses

	Parasites	Non-parasites	Statistic*	P
I. Females				
Relative frontal shield (mm)	-0.36 \pm 0.32 (25)	0.29 \pm 0.24 (36)	-1.64	0.11
Mass (g)	517.1 \pm 8.6 (26)	518.7 \pm 7.2 (35)	-0.14	0.88
Mass/tarsus (g mm ⁻¹)	8.23 \pm 0.12 (25)	8.15 \pm 0.10 (35)	0.53	0.60
Tarsus (mm)	63.2 \pm 0.30 (25)	63.6 \pm 0.34 (36)	-0.81	0.42
Age index (years)	1.87 \pm 0.14 (26)	1.49 \pm 0.12 (36)	-2.51	0.012
II. Males				
Relative frontal shield (mm)	-0.06 \pm 0.36 (23)	0.12 \pm 0.26 (34)	-0.41	0.68
Mass (g)	673.1 \pm 9.8 (24)	645.4 \pm 9.0 (34)	2.05	0.045
Mass/tarsus (g mm ⁻¹)	9.65 \pm 0.15 (24)	9.31 \pm 0.12 (34)	1.88	0.066
Tarsus (mm)	70.2 \pm 0.28 (23)	69.3 \pm 0.26 (34)	2.25	0.029
Age index (years)	1.90 \pm 0.14 (24)	1.60 \pm 0.11 (34)	-1.63	0.10

*Mann-Whitney *U*-test (*z*) for Age index, *t*-test for all other attributes.

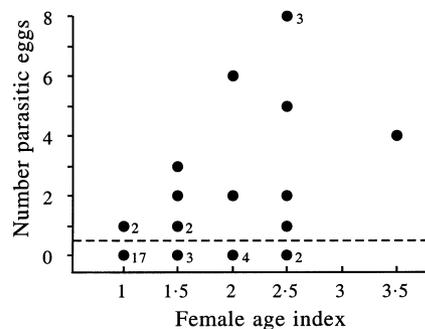


Fig. 3. The number of parasitic eggs laid by females at Pond S5 in relation to their age. Dashed horizontal line separates parasitic females (above) from non-parasitic females (below). Numbers beside points indicate multiple observations.

laid more parasitic eggs per host nest than younger females (Spearman correlation, $r_s = 0.60$, d.f. = 13, $P = 0.025$).

Comparing males, parasitic females' mates were larger than those of non-parasites in several measures: they were heavier, had larger tarsi and were in better condition (Table 3). Subsequent analysis showed that these patterns only held at Pond S5.

CHARACTERISTICS OF PARASITES RELATIVE TO HOSTS

The occurrence of parasitism appears to have been influenced by the body size of the potential parasitic female relative to the potential host female. Logistic regression revealed that parasitism was less likely to occur between dyads of females where the potential host was substantially heavier than the potential parasite (Wald $\chi^2 = 3.83$, $n = 88$ dyads, $P = 0.050$). Further analysis showed that this pattern was restricted to Pond S5 (Fig. 4; Wald $\chi^2 = 4.98$, $n = 43$ dyads, $P = 0.026$).

This relative size pattern could arise for two reasons: parasites might be larger, as a group, than hosts (absolute differences) or individual parasitic females might be larger than the individual hosts they parasitize (relative differences). Parasitic females were not, as a group, larger than host females (parasite mass 511.0 g (± 8.2) $n = 20$; host mass 524.4 g (± 9.0) $n = 24$, $t = 1.08$, $P = 0.29$; females that were both a host and a parasite were omitted). The above pattern thus appears to be due to relative differences between individual parasites and their hosts.

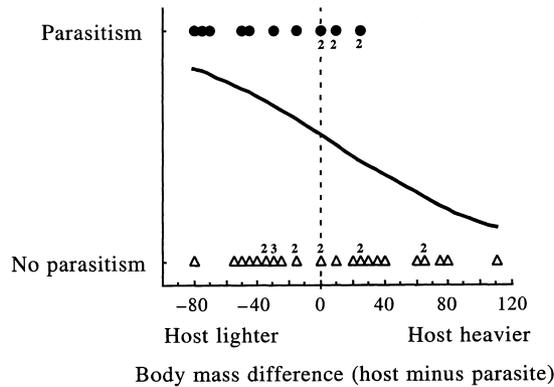


Fig. 4. The difference in body size between dyads of host and parasite females where parasitism occurred (filled circles) and between dyads of potential host and parasite females where parasitism did not occur (open triangles). Solid line is fitted logistic regression, and numbers indicate multiple samples with same values. Dashed line indicates equal size of females in the dyad. Parasitism was less likely to occur where hosts were substantially larger than parasites.

VARIATION IN THE BENEFITS OF ALLOCATING EGGS TO PARASITISM

To determine whether some females may have been constrained from parasitism by limited fecundity, I compared the clutch sizes of two classes of non-parasitic females that were likely to have been constrained from parasitism for different reasons: (i) females with no potential hosts, that were likely constrained by lack of hosts, and (ii) females with potential hosts, that were likely constrained from parasitism for other reasons, including low fecundity. Note that the clutch size that *parasitic* females lay in their own nests may not be a useful contrast for detecting fecundity limitation because the trade-offs between parasitism and nesting can favour a reduced parental clutch size for the brood parasites (Lyon 1998).

As predicted if low fecundity constrained some females from parasitism, the non-parasites with hosts laid significantly smaller clutches (7.68 ± 0.25 eggs, $n =$

84) than non-parasites without hosts (9.58 ± 0.19 eggs, $n = 25$; $t = 4.85$, $P < 0.0001$). These two groups of non-parasites did not differ in number of chicks fledged, which suggests that the difference in clutch size was due to differences in fecundity, not differences in the quality of the breeding situation (females with hosts, 3.78 chicks (± 0.22), $n = 50$; females without hosts, 4.05 chicks (± 0.35), $n = 19$; $t = 0.65$, $P = 0.52$). Seasonal effects, rather than fecundity differences, could account for the clutch size differences between the two types of non-parasites, because clutch size declines with laying date and the birds without hosts nested earlier, on average, than those with hosts. However, the difference in clutch size was also seen when the potential effects of season were controlled with analysis of covariance (Lyon 1998).

Contrary to the prediction that parasitism is associated with a low quality breeding situation, parasites did not lay smaller clutches than non-parasites with hosts, nor did they raise fewer chicks (Table 4). Furthermore, parasites did not have smaller or less vegetated territories (Table 4), nor did have lower risks of nest predation (Table 4).

Discussion

HOST AVAILABILITY AS A CONSTRAINT ON PARASITISM

Patterns of host availability indicate that some female coots were probably prevented from parasitic laying due to host limitation. The degree to which hosts are limiting in a given species will depend on the spatial and temporal patterns of host use that determine the pool of available hosts. In coots, most females laid parasitically only before initiating their own clutches and most parasitism involved immediate neighbours. With such a restricted spatial and temporal pattern of parasitism, it is not surprising to find evidence for host limitation. Depending on the spatial criterion used to define potential hosts, between 23% and 39% of the non-parasitic females had no hosts available when they

Table 4. Reproductive characteristics of parasites and non-parasites. Values are means \pm SE, with sample size in parentheses. Territory vegetation and size differed between the two wetlands, so are contrasted separately for each wetland

	Parasites	Non-parasites	Statistic*	P
Clutch size	7.89 \pm 0.25 (45)	7.68 \pm 0.19 (84)	$t = 0.66$	0.51
Chicks fledged (own nest)	4.04 \pm 0.28 (27)	3.78 \pm 0.22 (50)	$t = 0.74$	0.48
Nest predation rate†	19.5% (41)	19.8% (81)	$G = 0.001$	0.83
Territory vegetation index				
Pond S5	5.17 \pm 0.11 (31)	5.34 \pm 0.10 (56)	$z = 0.85$	0.39
Jaimeson	3.33 \pm 0.30 (12)	2.94 \pm 0.26 (21)	$z = 0.90$	0.36
Territory size (m)				
Pond S5	29.5 \pm 3.7 (16)	34.6 \pm 3.4 (18)	$t = 1.00$	0.32
Jaimeson	58.2 \pm 10.4 (4)	46.7 \pm 2.2 (15)	$t = 1.75$	0.10

* $z =$ Mann–Whitney U -test; $t = t$ -test; $G = G$ test of independence.

†Percentage of pairs that lost at least one clutch to predation.

began laying eggs and could have been constrained from parasitism due to host limitation.

By examining patterns of host availability for individual females, I have found clear evidence that host limitation affects the occurrence of brood parasitism in coots. Although host availability is thought to be an important factor in the evolution and maintenance of conspecific parasitism in general (Yom Tov 1980; Rohwer & Freeman 1989), most evidence for it is indirect and based on comparisons among populations or species. For example, the well known association between parasitism and high density nesting situations (Brown 1984; Semel & Sherman 1986; Rohwer & Freeman 1989) is thought to reflect the influence of host availability on brood parasitism. However, it could also be that in high nesting densities (i) parasites are better able to assess the value or accessibility of host nests (Emlen & Wrege 1986; Brown & Brown 1989) or (ii) competition is higher for limited nest sites, forcing females without nests to lay parasitically (Jones & Leopold 1967; Semel & Sherman 1986, 2001). It is also worth stressing that the assumption that higher densities of nests necessarily reflects higher densities of potential hosts will not always hold – the spatial scale of parasitism is crucial. For example, when parasitism occurs within an extremely limited spatial scale, as it does in coots, density will not influence host availability for individual females. Given these caveats, experiments will provide the clearest evidence for or against host limitation, but they are difficult to conduct. In an elegant experiment, Eadie (1991) altered the density of nest boxes in a population of goldeneye ducks (*Bucephala* spp.) and convincingly demonstrated that nest site limitation, not host availability, explained variation among lakes in the frequency of parasitism.

Parasitic females could have bypassed the constraints of host limitation simply by delaying breeding until after some of their neighbours had begun laying eggs. However, I found no evidence that parasites routinely did so, and parasitic females began egg-laying exactly at the same time as non-parasites, and not later, as would be expected if they had delayed breeding to wait for hosts. This finding indicates that parasitism is an opportunistic tactic that is employed by females who happen, by chance, to have hosts available when they are ready to begin laying eggs. Although brood parasites did not delay their first eggs to wait for hosts, brood parasitism nonetheless delayed initiation of their own nests relative to the nests of non-parasitic females, due to the time taken to lay parasitic eggs. Thus brood parasitism not only entails clutch size trade-offs for American coots (Lyon 1998) but trade-offs with timing of nest initiation as well.

Why don't females delay breeding to wait for hosts? One possibility is that the fitness costs of delaying breeding outweigh the benefits that would be gained from parasitism. One such cost is reduced success at renesting following nest predation. The probability of renesting decreased with nesting date (Fig. 5) and most

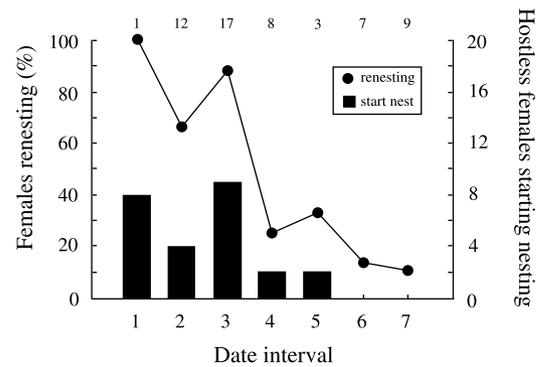


Fig. 5. The probability of successful renesting declines with date of first breeding, indicating a potential cost to waiting for hosts to become available. Circles show the percentage of all birds that began breeding in each four-day date interval from the start of the breeding season that successfully renested (i.e. raised ≥ 1 chick) after losing their first nest to predation. Numbers above circles indicate number of nests. Bars indicate the frequency distribution of first egg dates for all non-parasitic females that lacked hosts when they began breeding and would have had to delay nesting to wait for hosts to become available.

of the non-parasites without hosts were early nesting birds where successful renesting would have been likely (Fig. 5). Based on when their immediate neighbours began nesting, these females would have had to have waited at least 7 days for a host nest to become available (minimum estimate because nests are rarely parasitized as soon as the first host egg is laid), a delay that would have markedly decreased the probability of successful renesting (Fig. 5).

ACCESS TO HOST NESTS AS A CONSTRAINT ON PARASITISM

The opportunity to lay parasitically depends not only on the simple presence or absence of neighbouring nests to parasitize, but also on a female's ability to gain access to those nests. For most species, it is unclear how parasites gain access to host nests, but parasitism has been directly observed in a few species (Emlen & Wrege 1986; Brown & Brown 1989; McRae 1996).

In the absence of direct observations of parasitism, genetic or morphological comparisons can provide indirect clues about the role of cooperation or aggression in influencing access to host nests. I have shown elsewhere that parasitic female coots do not trade copulations for access to host nests (Lyon *et al.* 2002), ruling out host-parasite cooperation, so parasitic females gain access to host nest either by stealth or by aggression and dominance. Body size reliably determines the outcome of contests in many animals when the combatants differ sufficiently in size (Howard 1978; Austad 1983; Rubenstein 1984). In coots, both sexes defend the territory against neighbours, and females typically fight with females (unpublished data). Thus, the observation that parasitism was less likely to occur between dyads of females where the potential host

was substantially larger than the potential parasite certainly suggests that host dominance or nest defense prevents some females from laying parasitically. The observation that males at parasitized nests had smaller frontal shields than males at non-parasitized nests also suggests a role for host defense, given the clear social significance of frontal shields (Petrie 1988). Shield size has been directly linked to testosterone titre in American coots (Gullion 1953), thus it may be a reliable indication of an individual's strength and aggressiveness. However, further studies are needed to determine why host male shield size and body size differed between parasitized and unparasitized nests at only one of the two focal wetlands (Jaimeson Meadow).

An ability of some hosts to prevent their nests from being parasitized may also explain the positive correlation between the number potential hosts available to a female and the probability that she was parasitic (Fig. 1a). This correlation is not due simply to host availability (i.e. zero hosts vs. one or more hosts), because the correlation holds even when females without hosts are excluded from the analysis. Instead, it appears that increasing the pool of host nests available to a female increases the probability that she can gain access to at least one nest and be parasitic. Additional support for this idea is provided by a strong correlation between the number of hosts available to a brood parasite and the number of hosts actually used (Lyon 1993a).

VARIATION AMONG FEMALES IN THE BENEFITS OF ALLOCATING EGGS TO PARASITISM

I tested several hypotheses for why females might vary in their ability to benefit from allocating eggs to brood parasitism. I found no evidence that parasitic females had territories that were less prone to nest predation than non-parasites, thus enabling them to allocate eggs to parasitism rather than re-nesting. There was also no evidence that parasites had inferior breeding situations (smaller expected brood sizes, and therefore, smaller clutch sizes in their own nests) thus enabling them to allocate some of their limited eggs to parasitism. Finally, there was no evidence that parasites were delayed from breeding for reasons unconnected to parasitism, such as waiting for the vegetation to grow on their territories, and made use of the delay to engage in parasitism. Parasites did not differ in the amount of vegetation on their territories, a predictor of timing of breeding, and they did not begin laying eggs earlier in the season than non-parasites, as would be predicted by the delayed nesting hypothesis. Expanding the analysis of timing of breeding to all wetlands studied, not just the focal wetlands, did not alter this conclusion, nor did restricting the analysis to the subset birds on sparsely vegetated territories that nest late in the season (unpublished data).

Several observations do support the hypothesis that some females are constrained from parasitism due to low overall fecundity. First, the small clutches of non-parasitic females with hosts, relative to the clutch sizes of non-parasitic females without hosts, suggests that former were fecundity limited. An alternative explanation for the clutch size difference between these two groups of females – that they differed in breeding situation and resources available for chicks – is rejected because females in both groups fledged the same number of chicks. While it might seem puzzling that the observed clutch size difference between the two groups of females was not matched by a brood size difference, most females lay far bigger clutches than the number of chicks they can raise, and brood reduction through starvation is frequent (50% of all chicks starve, Lyon 1993a). Consequently, several of the eggs in each clutch are marginal eggs with relatively low survival rates that have small effects on the total number of chicks produced (Lyon 1993a, 1998).

The second indication that fecundity constraints prevent some females from laying parasitically or, conversely, that parasites are physiologically capable of laying more eggs than non-parasites, comes from the observations that parasitic females were older than non-parasites, that the total number of parasitic eggs laid by parasitic females increased with age, and that among parasites older females laid more eggs per host nest than younger females. The latter observation indicates that these patterns are due to age-dependent variation in fecundity rather than dominance effects, because dominance alone should not increase the number of successful visits to the same host nest. Sorenson (1991) also found age-dependent differences in brood parasitism and total fecundity in redhead ducks (*Aythya americana*). Age-dependent fecundity effects could arise from differences in foraging ability, territory quality or both (Alisauskas & Ankney 1985). In an Iowa population of coots, Crawford (1980) found that older females laid larger clutches in their own nests. Clutch size and age are not correlated in my population (unpublished data), probably because older birds engage in brood parasitism, which favours a reduced clutch size in the parasite's own nest (Lyon 1998).

Third, a comparison of parasitism rates on all of the wetlands I studied, not just the three focal wetlands, provides further evidence that limited fecundity may constrain some females from brood parasitism. The proportion of nesting females that engaged in brood parasitism on a wetland each year was negatively correlated with the average clutch size on the wetland (Fig. 6). This pattern holds when brood parasites are excluded from the calculation of mean clutch size on a wetland (Spearman correlation, $r_s = -0.68$, $P = 0.031$), so the result is not confounded by the smaller clutch sizes of parasites. One possible explanation for this negative correlation is that increases in the average optimal clutch size on a wetland decreases the proportion of females who have surplus fecundity to allocate

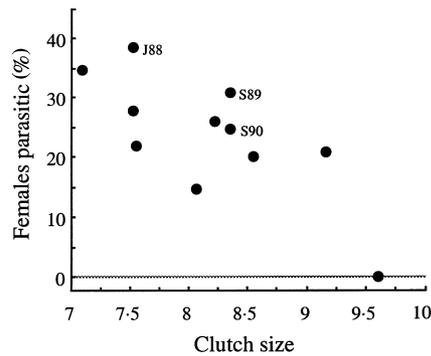


Fig. 6. The percentage of nesting females on a wetland that laid eggs parasitically decreased with the average clutch size on the wetland (Spearman rank correlation $r_s = -0.69$, $n = 11$ wetlands, $P = 0.028$). The three focal wetlands that are the focus of this study are identified; J88 is Jaimeson 1988, S89 is Pond S5 in 1989, and S90 is Pond S5 in 1990. The negative correlation remains when data for each wetland sampled in multiple years is pooled into a single overall average ($r_s = -0.75$, $n = 7$ wetlands, $P = 0.066$).

to parasitism. This assumes that all wetlands have the same distribution of female fecundities, but differ in the distribution of optimal clutch sizes determined by food availability for chicks (Lyon 1998). This is a reasonable assumption since the factors that limit egg production and chick production should differ because adults eat vegetable matter, while chicks are fed invertebrates (unpublished data). Nonetheless, the pattern shown here is correlational, and additional work is required to understand the underlying causal mechanisms.

Given the intriguing correlation between parasitism and clutch size (Fig. 6) it would be extremely interesting to examine the frequency of brood parasitism in the context of fecundity constraints in two populations of coots in Manitoba, Canada, that show dramatic differences in clutch size (6.6 at Delta Marsh, 9.6 at Minnedosa, Arnold 1990). Alisauskas & Ankney (1985) found that older females at Delta Marsh had higher protein reserves prior to breeding (Alisauskas & Ankney 1987), and they suggest that these reserves are important for clutch formation (but see Arnold & Ankney 1997). This could provide a physiological mechanism for the link I propose between age, fecundity and brood parasitism in coots.

VARIATION BETWEEN THE TWO FOCAL WETLANDS

Many of the correlates of parasitism I documented differed between the two focal wetlands. Attributes of host males differed from those of unparasitized males only at Jaimeson Meadow, while correlates with parasitic females were restricted to Pond S5. I cannot explain these differences without further study, but two general factors could be involved. First, these two wetlands showed consistent differences among years in factors that could affect parasitism, such as clutch size and fledging success. Second, I chose to study Jaimeson

Meadow as a focal wetland during a drought year when important reproductive parameters differed from those measured at the same wetland in other years: females were in worse condition (i.e. lighter, but not structurally smaller), nested in more marginal habitat and laid smaller clutches in 1988 than in other years at the same wetland (unpublished data). Distinguishing between these two hypotheses will be a fruitful avenue of future research.

FREQUENCY AND MAINTENANCE OF ALTERNATIVE TACTICS IN POPULATIONS

While my study was aimed at understanding why not all female coots engage in brood parasitism, my findings are also pertinent to a broader evolutionary question: what limits the frequency of alternative reproductive tactics in populations? Two general explanations could apply. First, the alternative tactic may be a component of behavioural plasticity, whereby individuals assess important conditional factors such as age, social status, or the opportunity to pursue the tactic, and then adjust their behaviour accordingly (West-Eberhard 1979; Dominey 1984; Repka & Gross 1995). Second, negative frequency-dependent selection might maintain the two alternatives as equal-fitness alternatives, either as a genetic polymorphism or with each individual investing the equilibrium investment in each tactic (a mixed evolutionarily stable strategy). The observation that parasitism by nesting female coots was correlated with several ecological and social factors indicates that it is a conditional tactic, not part of a stochastic mixed ESS (Austad 1984; Henson & Warner 1997).

Two considerations suggest that brood parasitism is likely to be a conditional strategy in most birds. First, conditional strategies enable organisms to finely tune their behaviour to match local environmental, social and physiological conditions (West-Eberhard 1979). For example, when females vary in fecundity, as I have shown for coots, not all females would benefit from a fixed allocation to parasitism, and a conditional strategy gives females the flexibility to make the appropriate allocation decision with their eggs. Second, although negative frequency-dependent fitness effects would, in theory, eventually limit the frequency of parasitism in the absence of other constraints, other factors are likely to constrain parasitism to a frequency below such an equilibrium frequency (Eadie & Fryxell 1992). The constraints I have shown for coots – host limitation and fecundity constraints – are likely to be important in most birds, so negative frequency-dependence alone is unlikely to determine the frequency of parasitism by nesting females in many avian populations.

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